cells proliferating to p68-86). Addition of recombinant IL-4 led to a significant decrease in IFN-γ which was still dependent upon antigenic stimulation (Figures 4A and 4C 0.19 ± 0.08 ng/ml without addition of MBP 68-86 versus 2.37 ± 0.8 in cells proliferating to p68-86, a 12 fold increase). Spleen T cells from anti-IGIF treated rats produced markedly reduced levels of IFN-γ in response to antigenic stimulation in cultures that were or were not supplemented with IL-4 (Figure 4A, 4.7 ± 0.4 ng/ml in spleen cells from anti-IGIF treated rat versus 9.7 ± 0.8 in spleen cells from rats treated with normal rabbit IgG and 13.5 ± 0.7 in PBS treated rats, with backgrounds of 0.4, 0.8 and 0.7, $p < 0.001$, when comparing anti-IGIF treatment to each control group). IL-4 production, however, markedly increased in splenic T cells from anti-IGIF treated rats regardless of *in vitro* stimulation (Figure 4B, 62.3 ± 4.2 pg/ml in spleen cells from anti-IGIF treated rat versus 15.3 ± 0.4 in spleen cells from rats treated with normal rabbit IgG and 15.6 ± 0.6 in PBS treated rats , $p < 0.001$, when comparing anti-IGIF treatment to each control group) unless cultures were supplemented with IL-4 (Figure 4D, 1860 ± 120 pg/ml in spleen cells from anti-IGIF treated rat versus 570 ± 30 in spleen cells from rats treated with normal rabbit IgG and 450 ± 35 in PBS treated rats, with backgrounds of 85, 42 and 34, $p < 0.0001$, when comparing anti-IGIF treatment to each control group). Addition of IL-4 to cultured spleen T cells (Figure 4C-D) did not exhibit a notable effect on their antigen specific proliferative response (data not shown).

TNF-α production was then evaluated in spleen cells from the above groups. The above spleen cells from anti-IGIF treated rats produced markedly reduced levels of TNF-α in response to antigenic stimulation (Figure 5, 850 ± 45 pg/ml in spleen cells from anti-IGIF treated rats versus 1975 ± 80 in spleen cells from rats treated with normal rabbit IgG and 2100 ± 110 in PBS treated rats, with backgrounds of 230, 210 and 270, respectively, $p < 0.001$, when comparing anti-IGIF treatment to each control group). Thus perturbation of the Th1/Th2 balance in anti-IGIF treated rats is associated with a marked reduction in TNF-α production.

Finally, the proliferative response of each group of cultured spleen cells to p68-86 was evaluated in a proliferation assay. (SI = 4.2 ± 0.3, 3.6 ± 0.4 and 3.96 ± 0.5 in spleen cells from rats treated with either anti-IGIF, normal rabbit IgG or PBS respectively). Thus anti-IGIF immunotherapy alters Th2/Th1 balance without a notable affect on antigen specific proliferative responsiveness.

Although the invention has been described in conjunction with specific embodiments thereof, it is evident that many alternatives, modifications and variations will be apparent to those skilled in the art. Accordingly, it is intended to embrace all such alternatives, modifications and variations that fall within the spirit and broad scope of the appended claims.

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